Shock in adults: Types, presentation, and diagnostic approach

INTRODUCTION — Shock is the physiologic state characterized by significant reduction of systemic tissue perfusion, resulting in decreased tissue oxygen delivery. This creates an imbalance between oxygen delivery and oxygen consumption. Prolonged oxygen deprivation leads to cellular hypoxia and derangement of critical biochemical processes at the cellular level, which can progress to the systemic level [1,2]:

- Cellular effects include cell membrane ion pump dysfunction, intracellular edema, leakage of intracellular contents into the extracellular space, and inadequate regulation of intracellular pH
- Systemic effects include alterations in the serum pH, endothelial dysfunction, and further stimulation of inflammatory and antiinflammatory cascades

The effects of oxygen deprivation are initially reversible, but rapidly become irreversible. The result is sequential cell death, end-organ damage, multi-system organ failure, and death. This highlights the importance of prompt recognition and reversal of shock [3].

The types and stages of shock are discussed in this topic review, as well as the clinical presentation and differential diagnosis of shock. A recommended diagnostic approach is also presented. Treatment of specific types of shock is discussed separately. (See "Management of severe sepsis and septic shock in adults" and see "Treatment and prognosis of cardiogenic shock complicating acute myocardial infarction" and see "Treatment of severe hypovolemia or hypovolemic shock in adults").

PHYSIOLOGY — Systemic tissue perfusion is determined by the cardiac output (CO) and systemic vascular resistance (SVR):
• CO is the product of heart rate and stroke volume. The stroke volume is related to preload, myocardial contractility, and afterload.

• SVR is governed by vessel length, blood viscosity, and the inverse of vessel diameter.

Decreased systemic tissue perfusion is a consequence of diminished CO or SVR. Both do not need to be decreased: Either can be elevated if the other is disproportionately low. As an example, SVR is decreased and CO is elevated in hyperdynamic shock [4]. In this setting, complex interactions between humoral and microcirculatory processes cause patchy regional blood flow and reduced effective tissue perfusion, resulting in derangement of cellular metabolic processes [5].

CO and SVR distinguish the different types shock, as described in the next section (show table 1).

TYPES OF SHOCK — Three types of shock states are recognized — hypovolemic, cardiogenic, and distributive.

Hypovolemic — Hypovolemic shock is a consequence of decreased preload due to intravascular volume loss. The decreased preload diminishes stroke volume, resulting in decreased cardiac output (CO). The systemic vascular resistance (SVR) is typically increased in an effort to compensate for the diminished CO and maintain perfusion to vital organs (show table 1).

Cardiogenic — Cardiogenic shock is a consequence of cardiac pump failure, resulting in decreased CO [3]. The SVR is typically increased in an effort to compensate for the diminished CO (show table 1).

Distributive — Distributive (vasodilatory) shock is a consequence of severely decreased SVR. The CO is typically increased in an effort to compensate for the diminished SVR (show table 1).

Combined — Different types of shock can coexist. As an example, patients with septic shock have a hypovolemic component due to decreased oral intake, insensible losses, vomiting, or diarrhea; a cardiogenic component due to sepsis-related myocardial dysfunction; and a distributive component due to activation of inflammatory and antiinflammatory cascades and their effects on vascular permeability and vasodilation.
STAGES OF SHOCK — Regardless of the type of shock, there exists a physiologic continuum. Shock begins with an inciting event, such as a focus of infection (eg, abscess) or an injury (eg, gunshot wound). This produces a systemic circulatory abnormality, which may progress through several complex and intertwined stages — preshock, shock, and end-organ dysfunction. This progression can culminate in irreversible end-organ damage and death [2,6].

Preshock — Preshock is also referred to as warm shock or compensated shock. It is characterized by rapid compensation for diminished tissue perfusion by various homeostatic mechanisms [7]. As an example, compensatory mechanisms during preshock may allow an otherwise healthy adult to be asymptomatic despite a 10 percent reduction in total effective blood volume. Tachycardia, peripheral vasoconstriction, and either a modest increase or decrease in systemic blood pressure may be the only clinical signs of shock [8].

Shock — During shock, the compensatory mechanisms become overwhelmed and signs and symptoms of organ dysfunction appear. These include tachycardia, dyspnea, restlessness, diaphoresis, metabolic acidosis, oliguria, and cool clammy skin.

The signs and symptoms of organ dysfunction typically correspond to a significant physiologic perturbation [6,9,10]. Examples include a 20 to 25 percent reduction in effective blood volume in hypovolemic shock, a fall in the cardiac index to less than 2.5 L/min/M2 in cardiogenic shock, or activation of innumerable mediators of the systemic inflammatory response syndrome (SIRS) in distributive shock [11].

End-organ dysfunction — Progressive end-organ dysfunction leads to irreversible organ damage and patient death. During this stage, urine output may decline further (culminating in anuria and acute renal failure), acidemia decreases the cardiac output and alters cellular metabolic processes, and restlessness evolves into agitation, obtundation, and coma.

CLINICAL PRESENTATION — Clinical presentation varies according to the type of shock and its cause. Several features are common among all types of shock (cardinal findings), while other features may suggest a particular type of shock (suggestive findings).

Cardinal findings — Cardinal features of shock include hypotension, oliguria, cool and clammy skin, abnormal mental status, and metabolic acidosis.

- Hypotension — Hypotension occurs in the majority of shock patients. It may be absolute hypotension (eg, systolic blood pressure <90 mmHg) or relative hypotension (eg, a drop in systolic blood pressure >40 mmHg). Relative
hypotension explains, in part, why a patient may be in shock despite having a high or normal blood pressure. Profound hypotension may occur, with vasopressors necessary to maintain adequate perfusion pressure as shock advances.

- Oliguria — Oliguria may be due to shunting of renal blood flow to other vital organs, intravascular volume depletion, or both. When intravascular volume depletion is a cause, it may be accompanied by orthostatic hypotension, poor skin turgor, absent axillary sweat, or dry mucous membranes. (See "Suggestive findings" below).

- Cool, clammy skin — Potent vasoconstrictive mechanisms compensate for decreased tissue perfusion by redirecting blood from the periphery to the vital organs, thereby maintaining coronary, cerebral, and splanchnic perfusion. This causes the cool and clammy skin that is typical of shock.

Not all patients with shock have cool and clammy skin, however. Patients with early distributive shock or terminal shock may have flushed, hyperemic skin. The former occurs prior to the onset of compensatory vasoconstriction, while the latter is due to failure of compensatory vasoconstriction.

- Change in mental status — The continuum of mental status changes frequently encountered in shock begins with agitation, progresses to confusion or delirium, and ends in obtundation or coma.

- Metabolic acidosis — Metabolic acidosis develops as shock progresses, reflecting decreased clearance of lactate by the liver, kidneys, and skeletal muscle [12]. Lactate production may increase due to anaerobic metabolism if shock progresses to circulatory failure and tissue hypoxia, which can worsen the acidemia. (See "Causes of lactic acidosis" and see "Arterial and mixed venous blood gases in lactic acidosis" and see "Simple and mixed acid-base disorders").

Suggestive findings — In addition to the above described cardinal symptoms and signs of shock, there may be additional findings from the history, physical examination, laboratory studies, or imaging that suggest a particular type of shock. These findings are neither sensitive nor specific.

- Hypovolemic shock — Depending on the cause of the hypovolemic shock, patients may report hematemesis, hematochezia, melena, vomiting, diarrhea, or abdominal pain. There may be evidence of blunt or penetrating trauma, or the patient may be postoperative. Physical manifestations may include decreased skin turgor (in younger patients), dry skin, dry axillae, dry tongue, or dry oral mucosa. In
addition, patients may have postural hypotension, decreased jugular venous pressure, or diminished central venous pressure. There may be anemia, or the amylase and lipase may be elevated. (See "Clinical manifestations and diagnosis of volume depletion in adults").

- **Cardiogenic shock** — Depending on the cause of the cardiogenic shock, patients may report dyspnea, chest pain, or palpitations. Lung examination may reveal diffuse crackles and cardiac examination may reveal a new murmur or soft heart sounds. The jugular venous pressure and central venous pressure may be increased. There may be evidence of pulmonary congestion or pulmonary edema on a chest radiograph, as well as recent or current ischemia on an electrocardiogram. Cardiac enzymes may be elevated. An echocardiogram may demonstrate the etiology. (See "Clinical manifestations and diagnosis of cardiogenic shock complicating acute myocardial infarction").

- **Distributive shock** — Depending on the cause of the distributive shock, patients may report dyspnea, productive cough, dysuria, hematuria, chills, myalgias, rashes, fatigue, malaise, headache, photophobia, pain, or a recent ingestion. There may be fever, tachypnea, tachycardia, leukocytosis, an abnormal mental status, or flushing. (See "Sepsis and the systemic inflammatory response syndrome: Definitions, epidemiology, and prognosis").

**DIFFERENTIAL DIAGNOSIS** — The differential diagnosis for the cause of shock is narrowed by determining which type of shock likely exists.

**Hypovolemic shock** — Hypovolemic shock can be divided into two categories, according to etiology:

- **Hemorrhage-induced** — Causes include blunt or penetrating trauma, upper or lower gastrointestinal bleeding, ruptured hematoma, hemorrhagic pancreatitis, fractures, or a ruptured aortic, abdominal, or left ventricular free wall aneurysm [13].

- **Fluid loss-induced** — Causes include diarrhea, vomiting, heat stroke, inadequate repletion of insensible losses, burns, and "third spacing". Third-space losses are common postoperatively and in patients who have intestinal obstruction, pancreatitis, or cirrhosis.

**Cardiogenic shock** — Causes of cardiac pump failure are diverse, but can be divided into four broad categories — myopathic, arrhythmic, mechanical, and extracardiac (obstructive).
Cardiomyopathies — Cardiomyopathic causes of shock include myocardial infarction involving greater than 40 percent of the left ventricular myocardium, right ventricular infarction, dilated cardiomyopathies, stunned myocardium following prolonged ischemia or cardiopulmonary bypass, and myocardial depression due to advanced septic shock [14].

Arrhythmias — Both atrial and ventricular arrhythmias can produce cardiogenic shock. Atrial fibrillation and atrial flutter reduce CO by interrupting coordinated atrial filling of the ventricles. Ventricular tachycardia, bradyarrhythmias, and complete heart block reduce CO, while ventricular fibrillation abolishes CO.

Mechanical abnormalities — Mechanical causes of cardiogenic shock include valvular defects, ventricular septal defects, atrial myxomas, and a ruptured ventricular free wall aneurysm. Valvular defects include rupture of a papillary muscle or chordae tendineae, acute aortic insufficiency caused by retrograde dissection of the ascending aorta into the aortic valve ring, and critical aortic stenosis. An atrial myxoma can reduce ventricular filling, while a ruptured left ventricular free wall aneurysm can produce pump failure, hypovolemia, and sudden death if is not contained by the pericardial sac.

Extracardiac abnormalities — Extracardiac (obstructive) causes of cardiogenic shock include massive pulmonary embolism, tension pneumothorax, severe constrictive pericarditis, pericardial tamponade, and severe pulmonary hypertension producing Eisenmenger's physiology. These conditions can also present clinically as hypovolemic shock when their primary physiologic disturbance is decreased preload, rather than pump failure. (See "Constrictive pericarditis" and see "Cardiac tamponade").

Distributive shock — There are many causes of distributive (vasodilatory) shock, most of which are discussed in detail separately.

- Septic shock (See "Sepsis and the systemic inflammatory response syndrome: Definitions, epidemiology, and prognosis" and see "Management of severe sepsis and septic shock in adults")

- Systemic inflammatory response syndrome (eg, pancreatitis, burns, multiple traumatic injuries) (See "Sepsis and the systemic inflammatory response syndrome: Definitions, epidemiology, and prognosis")

- Toxic shock syndrome (See "Epidemiology, clinical manifestations, and diagnosis of streptococcal toxic shock syndrome")
- Anaphylaxis and anaphylactoid reactions (See "Anaphylaxis: Rapid recognition and treatment")
- Drug or toxin reactions, including insect bites, transfusion reactions, and heavy metal poisoning (See "Use of blood products in the critically ill")
- Addisonian crisis, which should be considered if clinical signs of sepsis exist without evidence of infection [15] (See "Clinical manifestations of adrenal insufficiency in adults")
- Myxedema coma (See "Myxedema coma")
- Neurogenic shock after a central nervous system or spinal cord injury (See "Acute traumatic spinal cord injury")
- Acute systemic inflammation following acute myocardial infarction [16] (See "Clinical manifestations and diagnosis of cardiogenic shock complicating acute myocardial infarction", section on Hypotension and systemic vascular resistance)
- Post-resuscitation syndrome, which follows the return of spontaneous circulation after cardiac arrest [17]
- Post-cardiopulmonary bypass (see "Early noncardiac complications of coronary artery bypass graft surgery")

**RECOMMENDED APPROACH** — When a patient is suspected of having shock, diagnostic evaluation should occur at the same time as resuscitation. Resuscitative efforts should NOT be delayed for history, physical examination, laboratory testing, or imaging.

- Medical history — Some patients can provide a complete medical history despite profound shock, whereas others cannot provide any history. Historical data must instead be obtained from relatives or available medical records. The patient's baseline medical status, recent complaints, and recent activities may hold valuable information about the cause of shock. Additional clues include food and medicine allergies, recent changes in medications, potential acute or chronic drug intoxication, preexisting diseases, immunosuppressed states, and hypercoagulable conditions.
- Physical examination — Physical examination should be efficient and directed toward uncovering the severity, type, and cause of shock. Physical findings are neither sensitive nor specific for identifying the cause of shock.
Laboratory evaluation — Laboratory tests may help identify the cause of shock and early organ failure. They should be performed early in the evaluation of undifferentiated shock.

Potentially helpful laboratory tests include complete blood count with differential, basic chemistry tests (sodium, potassium, chloride, serum bicarbonate), blood urea nitrogen, creatinine, liver function tests, amylase, lipase, prothrombin time or international normalized ratio, partial thromboplastin time, fibrinogen, fibrin split products or dimer, cardiac enzymes (troponin or creatine phosphokinase isoenzymes), arterial blood gas, toxicology screen, and lactate level.

Venous whole blood lactate correlates with arterial lactate and can be used to screen for shock. In one observational study, an increasing lactate level predicted mortality among patients admitted to the hospital with suspected infection [18]. Increased serum lactate during shock results from increased production during anaerobic metabolism, mitochondrial derangements affecting oxygen utilization, and decreased clearance due to hepatic dysfunction.

A chest radiograph, abdominal radiograph for intestinal obstruction, abdominal computed tomography (CT), head CT scan, electrocardiogram, echocardiogram, or urinalysis may also be helpful. Gram stain of material from sites of possible infection (sputum, urine, wounds) may give early clues to the etiology of infection while cultures are incubating. Blood should be taken from two distinct venipuncture sites and inoculated into standard blood culture media [19].

With the data acquired from this evaluation, the cause of shock can usually be determined or narrowed to a few possibilities. For patients whose shock remains undifferentiated, pulmonary artery catheterization can provide additional information.

Pulmonary artery catheterization — Hemodynamic measurements obtained by pulmonary catheterization can be helpful in determining the type of shock that exists, particularly the cardiac output, pulmonary artery occlusion pressure (ie, pulmonary capillary wedge pressure), and systemic vascular pressure (show table 1). These measurements can also be used to guide fluid resuscitation, titrate vasopressors, and assess the hemodynamic effects of changes in mechanical ventilator settings (eg, positive end expiratory pressure [PEEP]) [20].

Despite these potential advantages, pulmonary arterial catheterization has never been shown to improve patient-important outcomes [21-23]. As a result, the clinical risks versus
benefits of pulmonary artery catheterization should be weighed on a patient-by-patient basis. (See "Swan-Ganz catheterization: Indications and complications").

Treatment of specific types of shock is discussed separately. (See "Management of severe sepsis and septic shock in adults" and see "Treatment and prognosis of cardiogenic shock complicating acute myocardial infarction" and see "Treatment of severe hypovolemia or hypovolemic shock in adults").

**MORTALITY** — Mortality due to shock is high. It is estimated that 35 to 60 percent of patients die within one month of the onset of septic shock [24]. The mortality rate may be even higher among patients with cardiogenic shock; it is estimated to be 60 to 90 percent [25,26]. Mortality due to hypovolemic shock is more variable. It depends upon the cause and the duration until recognition and treatment [7].

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**REFERENCES**


### Pathophysiology and hemodynamic profile of shock states

<table>
<thead>
<tr>
<th>Physiologic variable</th>
<th>Preload</th>
<th>Pump function</th>
<th>Afterload</th>
<th>Tissue perfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical measurement</td>
<td>Pulmonary capillary wedge pressure</td>
<td>Cardiac output</td>
<td>Systemic vascular resistance</td>
<td>Mixed venous oxygen saturation</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>↓</td>
<td></td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>↑</td>
<td></td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Distributive</td>
<td>↓ or ↔</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
</tbody>
</table>

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